1 Models of interacting particle systems in various fields

One of the objectives of this lecture is to build a bridge between probability theory and applications. We will start from various questions, mainly related to our environment or to industry issues, model them using random variables, and then use probability theory to study them.
This lecture does not aim to be exhaustive, as probabilistic models and applications are highly diverse, and there are numerous probabilistic methods for studying them. We will focus on certain models of interacting particles and on the concept of propagation of chaos. The term "particle" can refer to gas molecules, individuals, bacteria, animals, neurons, stock prices, and so on.

The terminology "propagation of chaos" comes from Kac and the idea is the following. We consider system of particles of size N that is initially chaotic, in the sense that particles are initially independent and identically distributed (i.i.d.), but then evolve through interaction. Thus, at each time, the particles are no more independent.
We say that there is propagation of chaos if, when we consider a fixed finite number of particles, and let the size of the system N go to infinity, those selected particles evolve independently in the limit, all with the same distribution. This implies that initially two particles are independent; then, due to the interaction, they become dependent, but when the size of the particle system is infinite, those two particles evolve independently ("chaotically"), according to a common distribution.

1.1 The McKean-Vlasov equation in physics

We start with the laboratory example described by Sznitman in his Saint Flour's lecture, entitled Topics in propagation of chaos, [Szn91].
The initial motivation for the subject was to try to investigate the connection between a detailed and a reduced description of particles' evolution, in a gas where interactions are pairwise such as in the Boltzmann equation and in the Landau equation.
The laboratory example proposed by Sznitmann is much simpler to study than the Boltzmann case. It is a model of interacting diffusions due to McKean. Let us consider a system of N particles in $\mathbb{R}^d$, with initial chaotic distribution $\mu_0^{\otimes N}$, satisfying the Stochastic Differential Equation (SDE)

$$\mathrm{d}X_t^{i,N} = \sqrt{2}\mathrm{d}B_t^i + \frac{1}{N}\sum_{j=1}^{N} b(X_t^{i,N}, X_t^{j,N})\mathrm{d}t \tag{1.1}$$

for $1 \leqslant i \leqslant N$, where $B = (B^1, \dots, B^N)$ is a standard N-dimensional Brownian motion and b is a regular compactly supported function (ensuring the existence and uniqueness of such a system). The particles evolve according to independent Brownian motions and interact through their drift terms. Introducing the empirical distribution $\overline{\mu}^N$ of the particle system: for $t \geqslant 0$,

$$\overline{\mu}_t^N = \frac{1}{N}\sum_{i=1}^{N} \delta_{X_t^{i,N}},$$

Equation (1.1) can be written

$$\mathrm{d}X_t^{i,N} = \sqrt{2}\mathrm{d}B_t^i + \int b(X_t^{i,N}, y)\mu_t^N(\mathrm{d}y).$$

Assuming that the initial particles are i.i.d. with a common distribution μ_0, as will be shown later, propagation of chaos holds as $N \to \infty$, and the common limiting distribution is the law of the solution, started from $X_0 \sim \mu_0$, to the *nonlinear* SDE

$$\mathrm{d}X_t = \sqrt{2}\mathrm{d}B_t + \int b(X_t, y)\mu_t(\mathrm{d}y)\mathrm{d}t,$$

where B is a standard one-dimensional Brownian motion and μ_t denotes the distribution of X_t. We say that the SDE is *nonlinear* because its coefficients depend on the distribution of the solution. If $\mu_0(\mathrm{d}x) = u_0(x)\mathrm{d}x$ has a density with respect to the Lebesgue measure, then one can prove that μ_t also admits a density $u(t, \cdot)$ and that u is a weak solution to the *nonlinear* Partial Differential Equation (PDE)

$$\begin{cases} \partial_t u = \Delta u - \mathrm{div}\big(\int b(x, y)u(t, y)\mathrm{d}y\, u\big) \\ u(0, \cdot) = u_0(\cdot), \end{cases}$$

where $u(t, x)$ is the density of the particles in position x at time t. This system of interacting particles has been the subject of several studies, and in Section 4 we will investigate the probabilistic approach developed in [BRTV98, Mal03].

1.2 Atlas model in finance

The Atlas model has been introduced by Fernholz, [Fer02], to model equity markets in finance. In the initial description of the model, the stock prices were modeled as Brownian motion except for the smaller stock price, which has an added positive drift that drives the market. Formally, let us consider $B = (B^1, \dots, B^N)$ a N-dimensional Brownian motion, the value of each stock price is solution to the SDE

$$\mathrm{d}X_t^{i,N} = \mathrm{d}B_t^i + b\mathbb{1}_{X_t^{i,N} = \min_{1 \leqslant k \leqslant N}\left(X_t^{k,N}\right)}\mathrm{d}t,$$

with b a positive real number. As in the myth of Atlas, the lowest process pushes the others up. Because the growth of the whole portfolio is supported only by this smallest stock, see Figure 1, the model is named after the Titan Atlas, eternally holding up the sky. These rank-based stochas-

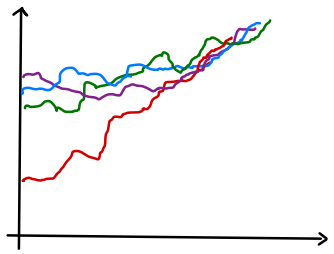

Figure 1: Illustration of trajectories of the Atlas model.

tic models successfully capture an empirical feature of real financial markets, namely the so-called

small-cap premium. More precisely, in rank-based financial models, each company is ordered according to its market capitalization. The dynamics of a stock then depend on its rank in the market. The phenomenon of small-cap premium refers to the observation that, over long time periods, stocks of smaller companies tend to have higher average returns than stocks of larger companies.

This model has then been generalized under the following form

$$\mathrm{d}X_t^{i,N} = \sum_{k=1}^{N} \sigma_k \mathbb{1}_{X_t^{i,N}=X_t^{(k)}} \mathrm{d}B_t^i + \sum_{k=1}^{N} b_k \mathbb{1}_{X_t^{i,N}=X_t^{(k)}} \mathrm{d}t \tag{1.2}$$

where $\sigma = (\sigma_1, \dots, \sigma_N) \in \mathbb{R}_+^N$, $b = (b_1, \dots, b_N) \in \mathbb{R}^N$, and $X^{(1)}, X^{(2)}, \dots, X^{(N)}$ are the order statistics of the N-tuple $(X^{1,N}, X^{2,N}, \dots, X^{N,N})$: for all $t \geqslant 0$,

$$\min_{1 \leqslant i \leqslant N} X_t^{i,N} = X_t^{(1)} \leqslant X_t^{(2)} \leqslant \ldots \leqslant X_t^{(N)} = \max_{1 \leqslant i \leqslant N} X_t^{i,N},$$

with i.i.d initial conditions $X_0^{1,N}, \dots, X_0^{N,N}$ of law μ_0. The existence and uniqueness of such a particle system have been studied in several articles. The weak existence and uniqueness when $\sigma \in (\mathbb{R}_+ \setminus \{0\})^N$ is a consequence of [BP87]. The strong existence and uniqueness were far more challenging to establish. They were first proved in the planar case ($N = 2$) in [FIKP13], and then extended to the high-dimensional setting up to the first triple collision in [IKS13], that is, before the time $\tau = \inf\Big\{t \geqslant 0 : \exists (i,j,k) \in \{1, \dots, N\}^3$ distinct such that $X_t^{i,N} = X_t^{j,N} = X_t^{k,N}\Big\}$. In [IKS13], a condition on σ is also provided to ensure that triple collisions cannot occur.

In two independent works, [JR13, KS18], propagation of chaos for the system (1.2) was proved under the following assumption:

> There exist $\sigma : [0,1] \to (0, +\infty)$ and $b : [0,1] \to \mathbb{R}$ continuous functions such that
>
> $$\sigma_k = \sigma\left(\frac{k}{N}\right) \quad \text{and} \quad b_k = b\left(\frac{k}{N}\right).$$

Introducing the empirical cumulative distribution

$$F_t^N(x) = \frac{1}{N} \sum_{k=1}^{N} \mathbb{1}_{\{X_t^{k,N} \leqslant x\}},$$

we observe that Equation (1.2) can be written as

$$\mathrm{d}X_t^{i,N} = \sigma\Big(F_t^N(X_t^{i,N})\Big)\mathrm{d}B_t^i + b\Big(F_t^N(X_t^{i,N})\Big)\mathrm{d}t.$$

There is propagation of chaos when $N \to \infty$, and the common distribution is the law of the solution starting from $X_0 \sim \mu_0$ to the *nonlinear* SDE

$$\mathrm{d}X_t = \sigma(F_t(X_t))\mathrm{d}B_t + b(F_t(X_t))\mathrm{d}t,$$

where B is a standard one-dimensional Brownian motion and $F_t(x) = \mathbb{P}(X_t \leqslant x)$ is the cumulative distribution function of X_t. It was also proved that the cumulative distribution function $F(t, \cdot) :=$

$F_t(\cdot)$ of X_t is the weak solution of the nonlinear PDE

$$\begin{cases} \partial_t F = \frac{1}{2}\partial_x^2(A(F)) - \partial_x(B(F)) \\ F(0,x) = F_0(x) \end{cases}$$

where $A(u) = \int_0^u \sigma^2(v)\mathrm{d}v$, $B(u) = \int_0^u b(v)\mathrm{d}v$, and $F_0(x) = \mu_0((-\infty, x])$.

1.3 Neural dynamics in neurosciences

The neurons in our nervous system communicate with each other through an electrical signal that propagates along their axons. This specific electrical signal is characterized by rapid depolarization (spike), followed by hyperpolarization, before returning to the resting membrane potential.

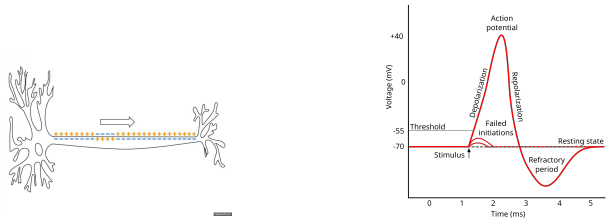

Figure 2: Action potential of a neuron (Source: Wikipedia).

We present the model introduced in [DMGLP15], which describes the time evolution of a population of N interacting neurons. Each neuron spikes randomly according to a point process whose rate depends on its membrane potential.
For $i \in \{1, \dots, N\}$, $X_t^{i,N}$ models the membrane potential of neuron i at time t. Neurons interact either by electrical, or by chemical synapses:

- Electrical synapses: they occur through gap-junctions which allow neurons in the brain to communicate directly. This induces an attraction between the values of the membrane potentials and, thus a deterministic drift of the system towards its average potential.
- Chemical synapses: each neuron spikes randomly following a point process with rate depending on the membrane potential of the neuron. At its spiking time, the membrane potential of the spiking neuron is reset to the value 0. At the same time, simultaneously, the other neurons, which do not spike, receive an additional amount of potential $\frac{1}{N}$, which is added to their membrane potential.

The generator of the particle system described above is thus given by, for any smooth test function $\varphi : \mathbb{R}_+^N \to \mathbb{R}$,

$$\mathcal{L}\varphi(x) = -\lambda \sum_{i=1}^N \left(\frac{\partial \varphi}{\partial x_i}(x)(x_i - \overline{x}) \right) + \sum_{i=1}^N f(x_i)\big(\varphi(x + \delta^i(x)) - \varphi(x)\big),$$

where $\delta^i(x) = (\delta_j^i(x))_{1 \leqslant j \leqslant N}$ with $\delta_j^i = \begin{cases} \frac{1}{N} & \text{if } j \neq i \\ -x_i & \text{if } j = i \end{cases}$, and $\overline{x} = \dfrac{1}{N}\displaystyle\sum_{i=1}^N x_i$. The quantities $\lambda \geqslant 0$ and the function f, called the spiking rate of the system, are parameters of the system.

The first term of the generator $\mathcal{L}$ describes the attraction of the neurons to the mean potential, and the second term describes jumps occurring at rate $f(x_i)$ due to the spiking of neurons with potential x_i. We assume that the function f is non-decreasing to model the phenomenon that the higher the potential, the more likely it is to jump to 0.
The evolution in time of the potential of each neuron can be also be described by a system of SDE driven by Poisson measures. Let $\left(Q^i(\mathrm{d}s,\mathrm{d}z)\right)_{i\geqslant N}$ be independent Poisson measures on $\mathbb{R}_+\times\mathbb{R}_+$ with intensity $\mathrm{d}s\mathrm{d}z$ modeling the spikes, and $(X_0^{i,N})_{1\leqslant i\leqslant N}$ be a sequence of i.i.d initial potentials, independent of the Poisson measures. Consequently, we write

$$\begin{aligned}X_t^{i,N} =& X_0^{i,N} - \lambda\int_0^t \left(X_s^{i,N} - \overline{X}_s^N\right)\mathrm{d}s \\ &- \int_0^t\int_{\mathbb{R}_+} X_{s-}^{i,N}\mathbb{1}_{\left\{z\leqslant f(X_{s-}^{i,N})\right\}}Q^i(\mathrm{d}s,\mathrm{d}z) + \frac{1}{N}\sum_{j\neq i}\int_0^t\int_{\mathbb{R}_+}\mathbb{1}_{\left\{z\leqslant f(X_{s-}^{j,N})\right\}}Q^j(\mathrm{d}s,\mathrm{d}z),\end{aligned}$$

where $\overline{X}_t^N = \frac{1}{N}\sum_{i=1}^N X_t^{i,N}$ is the mean potential of the system.
The asymptotic behavior this system is studied in [DMGLP15, FL16], and they state the propagation of chaos to the distribution of the solution X to the following nonlinear SDE

$$X_t = X_0 - \lambda\int_0^t (X_s - \mathbb{E}[X_s])\mathrm{d}s - \int_0^t\int_{\mathbb{R}_+} X_{s-}\mathbb{1}_{\{z\leqslant f(X_{s-})\}}Q(\mathrm{d}s,\mathrm{d}z) + \int_0^t \mathbb{E}[f(X_s)]\mathrm{d}s,$$

where $Q(\mathrm{d}s,\mathrm{d}z)$ is a Poisson measure on $\mathbb{R}_+\times\mathbb{R}_+$ with intensity $\mathrm{d}s\mathrm{d}z$.
From the PDE point of view, the density function $u(t,\cdot)$ of the distribution of X_t ($u(x,t)$ represents the density of neurons with membrane potential x at time t) satisfies the following equation:

$$\begin{cases}\partial_t u = (\lambda x - a_t)\partial_x u + (\lambda - f(x))u \\ u(t,0) = \frac{p_t}{a_t} \\ u(0,x) = u_0(x)\end{cases}$$

where $a_t = \int_0^\infty (f(x)+\lambda x)u(t,x)\mathrm{d}x$, $p_t = \int_0^\infty f(x)u(t,x)\mathrm{d}x$, and u_0 denotes the initial distribution. We study this system and its long lime behavior in detail in Section 5.

1.4 Epidemiologic models in medecine

We consider individual-based SIRS-type epidemiological models describing the spread of an infectious disease in which individuals can become infected, recover, and subsequently return to the susceptible state without acquiring total immunity. In such a population, susceptible individuals become infected through contact with infected individuals, while infected individuals recover at a given rate and immediately become susceptible again. We consider a closed population of size N (without immigration).
We assume that the interactions in the population are uniform between the individuals, and all the individuals behave in the same way to the disease.

Each individual is characterized by two random quantities that evolve over time: their infectivity, which determines the rate at which they transmit the disease, and their susceptibility, corresponding to the probability of being reinfected upon contact with an infected individual. The infectivity of

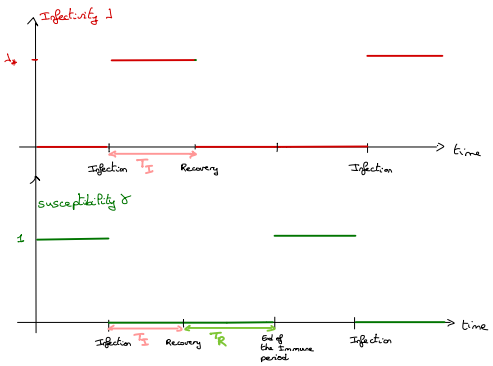

Figure 3: Evolution in time of the infectivity and susceptibility of an individual.

individual $i \in \{1, \dots, N\}$ at time t is denoted by $\lambda^{i,N}(t)$, while their susceptibility is denoted by $\gamma^{i,N}(t)$. Their value depends on the age $a^i(t)$ of individual i, namely the elapsed time since the last infection.

Each individual repeatedly alternates between two states: an infectious state (denoted by I), in which the individual cannot be infected but can spread the disease ($\lambda^{i,N}(t) \geqslant 0$ and $\gamma^{i,N}(t) = 0$), and a susceptible state (denoted by S), in which the individual does not spread the disease but can be reinfected ($\lambda^{i,N}(t) = 0$ and $\gamma^{i,N}(t) \geqslant 0$).

The individuals interact through the global infection $\mathfrak{F}^N(t)$ of the disease: individual i becomes infected at time t at rate $\gamma^{i,N}(t)\mathfrak{F}^N(t)$, where $\mathfrak{F}^N(t)$ is given by

$$\mathfrak{F}^N(t) = \frac{1}{N} \sum_{k=1}^{N} \lambda^{k,N}(t).$$

At each new infection, new infectivity and susceptibility curves are sampled for the infected individual, and its age is reset to 0, while the other individuals keep their own infectivity and susceptibility curves, as well as their ages.

In the classical SIRS model, the functions λ and γ are indicator functions of the age a since the last infection:

$$\lambda(a) = \lambda_* \mathbb{1}_{a \leqslant T_I}, \tag{1.3}$$

where T_I is the *random* duration of the infectious period and λ_* is a positive constant, and

$$\gamma(a) = \mathbb{1}_{a > T_R + T_I}, \tag{1.4}$$

where T_R is the *random* length of the immune period. Consequently, after recovery, the individual remains immune to the disease for a period of length T_R (and therefore cannot be infected), before becoming fully susceptible to the disease again (see Figure 3). The SIS model corresponds to the case where $T_R = 0$ almost surely. Models with waning immunity have recently been introduced and studied in [FPPZN25, GZN25].

■ **Exercise 1.** Consider a SIRS model for a population of size N.

The infectivity and susceptibility curves are given by (1.3) and (1.4), respectively. We assume that the *generic* distribution of the pair (T_I, T_R) admits a density f on $\mathbb{R}_+ \times \mathbb{R}_+$. Note that, in the

classical SIRS model, T_I and T_R are independent exponential random variables with respective rates $\nu > 0$ and $\delta > 0$.
The aim of this exercise is to write the system of SDEs describing the evolution of the disease, more precisely the infectious state of each individual. To this end, we introduce, for each individual, its age, infectious period, and immune period. For $i \in \{1, \dots, N\}$, $a^i(t)$ denotes the age of individual i at time $t \geqslant 0$, and $T^i(t) = (T_I^i(t), T_R^i(t))$ denotes the pair of infectious and immune durations associated with individual i at time t.
Let $a^i(0)$ and $T^i(0) = (T_I^i(0), T_R^i(0))$ denote their initial values.

1. Describe the evolution in time of $(a^i, T^i)_{1 \leqslant i \leqslant N}$ in your words.
2. Introducing independent Poisson measures $\left(Q^i\right)_{1 \leqslant i \leqslant N}$, write a system of SDEs and/or the infinitesimal generator satisfied by $(a^i, T^i)_{1 \leqslant i \leqslant N}$.
3. Without proof, give the SDE and/or the generator satisfied by the limiting distribution as the population size tends to infinity ($N \to \infty$).

1.5 A few interesting questions on such systems

One of the main objectives in the study of such systems is to analyze their long time behavior, that is, their behavior as $t \to \infty$: is there existence of an equilibrium in long time? For example, in epidemiology, a central question is to identify conditions ensuring the existence of an endemic equilibrium, that is, a regime in which the disease persists in the population (something that public health policies usually aim to prevent).
For a system of i.i.d. particles, studying a single particle is sufficient to characterize the long time behavior of the whole system. In contrast, when the particles interact, the analysis of the long time behavior becomes significantly more challenging, since the behavior of each particle is influenced by that of the others. Nevertheless, when propagation of chaos holds, the study of a large population can be reduced to the analysis of a *typical* particle, namely the solution of a nonlinear equation describing the limiting dynamics as the population size tends to infinity. This reduction is one of the main strengths of the propagation of chaos property.